#17 Nucleotide re: Rule

PATENT

Docket No.: **19603/3340 (CRF D-2018B)**

IN THE UNITED STATES PATENT AND TRADEMARK OFFICE

Applicants	: Qiu et al.	**ATTENTION: DIRECTOR TC 1600**
Serial No.	: 09/597,840	
Cnfrm. No.	: 6516	Examiner: A. Kubelik
Filed	: June 20, 2000	
For	: ENHANCEMENT OF GROWTH IN PLANTS	Art Unit: 1638

RENEWED PETITION UNDER 37 C.F.R. § 1.144 FOR REVIEW OF RESTRICTION REQUIREMENT

U.S. Patent and Trademark Office
Crystal Mall 1
1911 South Clark Place, 7th Floor
Arlington, Virginia 22202
ATTENTION: DIRECTOR TC 1600

Dear Center Director:

In response to the Petition Decision of November 22, 2002 ("Petition Decision"), applicants hereby renew their petition pursuant to 37 C.F.R. § 1.144 for withdrawal of the September 7, 2001, restriction requirement.

Claims 38-51 are currently pending before the U.S. Patent and Trademark Office ("PTO"). Claim 38 is an independent claim directed to a method of enhancing growth in plants compared to untransformed plants or plant seeds by providing a transgenic plant or plant seed transformed with a DNA molecule encoding a hypersensitive response elicitor protein or polypeptide and growing the transgenic plants or transgenic plants produced from the transgenic plant seeds under conditions effective to enhance plant growth. The remaining claims each depend (directly or indirectly) from claim 38. The particular claims at issue in the restriction requirement are claims 40, 42-45, and 51. Claims 40 and 42-45 recite different pathogen sources of the hypersensitive response elicitors used in the claimed methods. These sources include *Erwinia chysanthemi* (claim 40), *Pseudomonas syringae* (claim 42), *Pseudomonas solanacearum* (claim 43), *Xanthomonas campestris* (claim 44), and a *Phytophthora* species (claim 45). Claim 51 adds the limitation of "applying the

hypersensitive response elicitor polypeptide or protein to the propagated plants to enhance growth of the plant." Thus, the claims of the present application are directed to a method of using plants transformed with DNA molecules encoding hypersensitive response elicitors; they are not directed to hypersensitive response elicitors themselves which were known to exist at the time the present invention was made.

In issuing its Petition Decision, the PTO cites Manual of Patent Examining Procedure ("MPEP") § 803.04 which states that "[a]bsent evidence to the contrary, each such nucleotide sequence is presumed to present an independent and distinct invention." The PTO has also taken the position that the relevant art is unpredictable and, therefore, that the skilled artisan would not predict that the results obtained with one elicitor protein would be obtained with all the others. Applicants respectfully disagree.

On its face, MPEP § 803.04 sets forth a rebuttable presumption that nucleotide sequences encoding different proteins are independent and distinct inventions subject to restriction. Applicants respectfully submit that the evidence and argument presented in its Petition Under 37 C.F.R. § 1.144 for Review of Restriction Requirement, dated September 27, 2002, as well as the new evidence provided herein, are more than sufficient to overcome this presumption. In particular, applicants submit herewith a Supplemental Declaration of Zhong-Min Wei Under 37 C.F.R. § 1.132 ("Supplemental Wei Declaration"), which includes additional evidence showing that hypersensitive response elicitors from a diverse range of plant pathogens (1) are an art-recognized class of proteins where results achieved with one such protein would be expected when other proteins in this class are used and (2) share the unique ability to cause distinct plant responses.

In plants, the hypersensitive response phenomenon results from an incompatible interaction between plant pathogens and non-host plants (Supplemental Wei Declaration ¶ 5). As explained in Gopalan et al., "Bacterial Genes Involved in the Elicitation of Hypersensitive Response and Pathogenesis," Plant Disease 80: 604-10 (1996) ("Gopalan") (attached to Supplemental Wei Declaration as **Exhibit 1**), these types of interactions involve, for example, a bacterial plant pathogen attempting to infect a host plant, and the host plant preventing proliferation of the pathogen by the collapse and death, or necrosis, of plant leaf cells at the site of infection (Id.). This is distinct from a compatible interaction between a bacterial plant pathogen and a host plant in which the bacteria is capable of proliferation, resulting in the spread of the pathogen throughout the plant and the manifestation of disease symptoms. Gopalan at 604 (Id.).

Hypersensitive response elicitors within a given genus are often homologous to elicitors from different pathogenic species and strains of the same genus (Supplemental Wei Declaration ¶ 6). For example, homologs of hypersensitive response elicitors from *Erwinia amylovora* and *Pseudomonas syringae* have been identified in different bacteria species and strains from the genera *Erwinia* and *Pseudomonas*, respectively. See Gopalan (Id.).

In addition, numerous reported studies confirm that a gene encoding a hypersensitive response elicitor from a particular source genus can be used to isolate a corresponding hypersensitive response elicitor gene from different species and strains of that same genus (Supplemental Wei Declaration ¶ 7). For example, in Bauer et al., "*Erwinia chrysanthemi* $Harpin_{Ech}$: An Elicitor of the Hypersensitive Response that Contributes to Soft-Rot Pathogenesis," MPMI 8(4): 484-91 (1995) ("Bauer") (attached to Supplemental Wei Declaration as **Exhibit 2**), the *Erwinia amylovora* hypersensitive response elicitor encoding gene was used as a probe to isolate, clone, and sequence the gene encoding the *Erwinia chrysanthemi* hypersensitive response elicitor, as follows:

> The cosmids were probed in colony blots with a 1.3-kb *Hind*III fragment from pCPP1084, which contains the *E. amylovora hrpN* gene (Wei et al. [, "Harpin Elicitor of the Hypersensitive Response Produced by the Plant Pathogen *Erwinia amylovora*," Science 257:85-88 (]1992[)]). pCPP2157, one of the three cosmids hybridizing with the probe, was digested with several restriction enzymes, and the location of the $hrpN_{Ech}$ gene in those fragments was determined by probing a Southern blot with *E. amylovora Hind*III fragment. Two fragments, each containing the entire $hrpN_{Ech}$ gene, were subcloned into different vectors: pCPP2142 contained an 8.3-kb *Sal*I fragment in pUC119 (Vieira and Messing [,"Production of Single-Stranded Plasmid DNA," Methods Enzymol., 153:3-11(] 1987[)]), and pCPP2141 contained a 3.1-kb *Pst*I fragment in pBluescript II SK(-) (Stratagene, La Jolla, CA).
>
> ***Sequence of $hrpN_{Ech}$***
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> The nucleotide sequence of a 2.4-kb region of pCPP2141 encompassing $hrpN_{Ech}$ was determined. The portion of that sequence extending from the putative ribosome-binding site through the $hrpN_{Ech}$ coding sequence to a putative rho-independent terminator is presented in Figure 1.

Bauer at 485 (Id.).

In the same manner as described in Bauer *supra*, Cui et al., "The RsmA$^-$ Mutants of *Erwinia carotovora* subsp. *carotovora* Strain Ecc71 Overexpress *hrp*N_{Ecc} and Elicit a Hypersensitive Reaction-like Response in Tobacco Leaves," MPMI 9(7): 565-73 (1996) ("Cui") (attached to Supplemental Wei Declaration as **Exhibit 3**) demonstrates that the gene encoding the *Erwinia carotovora* hypersensitive response elicitor can be isolated, sequenced, and cloned by using the *Erwinia chrysanthemi* hypersensitive response elicitor encoding gene to probe the genomic library of *Erwinia carotovora* (Supplemental Wei Declaration ¶ 8). Further, Cui states the following:

> The genomic library of *E. carotovora* subsp. *carotovora* strain Ecc71 in pLARF5 was screened by in situ colony hybridization with a 0.75-kb internal *Cla*I fragment of *hrpN* of *E. chrysanthemi* (Bauer et al.[, "*Erwinia chrysanthemi* $Harpin_{Ech}$: An Elicitor of the Hypersensitive Response that Contributes to Soft-Rot Pathogenesis," MPMI 8(4): 484-91 (]1995). Two cosmids, pAKC921 and pAKC922, that hybridized with the probe were isolated. The subclones (pAKC923 and pAKC924, Table 1) carrying *hrpN* DNA were used for sequence analysis.

Cui at 572 (Id.).

The gene encoding the hypersensitive response elicitor of *Erwinia amylovora* has also been used as a probe to isolate and clone the gene encoding the hypersensitive response elicitor of *Erwinia stewartii* (Supplemental Wei Declaration ¶ 9). It was found that antibodies raised against the hypersensitive response elicitor of *Erwinia stewartii* cross-reacted with the hypersensitive response elicitor of *Erwinia amylovora*. See Ahmad et al., "Harpin Is Not Necessary for the Pathogenicity of Maize," 8th Int'l Cong. Molec. Plant Microbe Inter. July 14-19, 1996 ("Ahmad") (attached to Supplemental Wei Declaration as **Exhibit 4**) (Id.).

Similar findings were reported for hypersensitive response elicitors from the genus *Pseudomonas* (Supplemental Wei Declaration ¶ 10). An internal fragment of the hypersensitive response elicitor from *Pseudomonas syringae* pv. *syringae* (i.e., *hrpZ*) was used to identify and isolate the hypersensitive response elicitors from *P. syringae* pv. *glycinea* and *P. syringae* pv. *tomato* (Id.). Significant amino acid sequence similarities were identified between the various *Pseudomonas syringae* elicitors. See Preston et al., "The HrpZ Proteins of *Pseudomonas syringae* pvs. *syringae, glycinea,* and *tomato* Are Encoded by an Operon Containing *Yersinia ysc* Homologs and Elicit the Hypersensitive Response in Tomato But Not Soybean," MPMI 8(5): 717-32 (1995) ("Preston") (attached to Supplemental Wei Declaration as **Exhibit 5**) (Id.).

The genes encoding hypersensitive response elicitors are positioned within the *hrp* gene cluster or proximate to the *hrp* gene cluster in *hrp* regulons (Supplemental Wei Declaration ¶ 11). For example, *hrpN* from *Erwinia amylovora* was located within the *hrp* gene cluster, as was *hrpZ* from *Pseudomonas syringae* (Id.). The *popA* gene, encoding a hypersensitive response elicitor from *Pseudomonas solanacearum*, was located on the left flank of the *hrp* gene cluster within a *hrp* regulon. See Bonas, "*hrp* Genes of Phytopathogneic Bacteria," Current Topics in Microbiology and Immunology 192: 79-98 (1994) ("Bonas I") (attached to Supplemental Wei Declaration as **Exhibit 6**) and Alfano et al., "The Type III (Hrp) Secretion Pathway of Plant Pathogenic Bacteria: Trafficking Harpins, Avr Proteins, and Death," Journal of Bacteriology 179: 5655-5662 (1997) ("Alfano") (attached to Supplemental Wei Declaration as **Exhibit 7**) (Id.). Similar to the *popA* gene, *hreX*, the gene encoding the hypersensitive response elicitor from *Xanthomonas campestris*, was located on the left flank of the *hrp* gene cluster. See Swanson et al., "Isolation of the *hreX* Gene Encoding the HR Elicitor Harpin (Xcp) from *Xanthomonas Campestris* pv. *pelargonii*," Phytopathology 90: s75 (1999) ("Swanson") (attached to Supplemental Wei Declaration as **Exhibit 8**) (Id.).

The characteristics that distinguish hypersensitive response elicitors as a distinct class of molecules are clearly apparent when considering the different elicitors' secretion mechanisms, regulation, biochemical characteristics, and biological activities (Supplemental Wei Declaration ¶ 12).

Substantially all hypersensitive response elicitors identified have been shown to be secreted through the type III, *hrp* dependent secretion pathway (Supplemental Wei Declaration ¶ 13). The type III secretion pathway is a highly conserved and unique mechanism for the delivery of pathogenicity related molecules in gram-negative bacteria (Id.). The *hrp* gene cluster is largely composed of components of the type III secretion system. See Bogdanove et al., "Unified Nomenclature for Broadly Conserved *hrp* Genes of Phytopathogenic Bacteria," Molec. Microbiol. 20:681-83 (1996) ("Bogdanove") (attached to Supplemental Wei Declaration as **Exhibit 9**); and Alfano (Id.).

Regulation of the genes encoding the *hrp* gene cluster, and subsequently the genes encoding the components of the type III secretion system and hypersensitive response elicitors, is controlled by environmental factors (Supplemental Wei Declaration ¶ 14). Specifically, transcriptional expression of these genes is induced under conditions that mimic the plant apoplast, such as low concentrations of carbon and nitrogen, low temperature, and low pH. See Wei et al., "Regulation of hrp Genes and Type III Protein Secretion in *Erwinia amylovora* by HrpX/HrpY, a Novel Two-Component System, and HrpS," MPMI 13(11):

1251-1262 (2000) ("Wei I") (attached to Supplemental Wei Declaration as **Exhibit 10**); and Bonas I (Id.).

Biochemically, hypersensitive response elicitors have a number of common characteristics (Supplemental Wei Declaration ¶ 15). These include being glycine rich, heat stable, hydrophilic, lacking of an N-terminal signal sequence, and susceptible to proteolysis. See Bonas, "Bacterial Home Goal by Harpins," Trends Microbiol 2: 1-2 (1994) ("Bonas II") (attached to Supplemental Wei Declaration as **Exhibit 11**); Bonas I; Gopalan; and Alfano (Id.).

In addition, hypersensitive response elicitors share a unique secondary structure that has been associated with these elicitors' distinct biological activities (described below) (Supplemental Wei Declaration ¶ 16). The structure has two primary components, an alpha helix unit and a relaxed acidic unit having a sheet or random turn structure (Id.). In the absence of one or both of these components, hypersensitive response elicitation does not occur. See WO 01/98501 to Fan et al. ("Fan") (attached to Supplemental Wei Declaration as **Exhibit 12**) (Id.).

In addition to eliciting the hypersensitive response in a broad range of plant species, as explained by Wei et al., "Harpin from *Erwinia amylovora* Induced Plant Resistance," Acta Horticulture 411: 223-225 (1996) ("Wei II") (attached to Supplemental Wei Declaration as **Exhibit 13**) and by Alfano, hypersensitive response elicitors also share the ability to induce specific plant responses (Supplemental Wei Declaration ¶ 17). The induction of plant disease resistance, plant growth enhancement, and plant stress resistance are three plant responses that result from treatment of plants or plant seeds with a hypersensitive response elicitor from a gram-negative plant pathogen (Id.).

As described in Wei II, treatment of plants with the hypersensitive response elicitor HrpN from *Erwinia amylovora* resulted in disease resistance to a broad range of plant pathogens (Supplemental Wei Declaration ¶ 18). For example, HrpN induced disease resistance to southern bacterial wilt (*Pseudomonas solanacearum*) in tomato, tobacco mosaic virus in tobacco, and bacterial leaf spot (*Gliocladium cucurbitae*) in cucumber (Id.).

The hypersensitive response elicitor HrpZ from *Pseudomonas syringae* was reported to induce disease resistance in cucumber to a diverse range of pathogens, including the fungal disease *Colletotrichum lagenarium*, tobacco necrosis virus, and bacterial angular leaf spot (*Pseudomonas syringae* pv. *lachrymans*). See Strobel et al., "Induction of Systemic Acquired Resistance in Cucumber by *Pseudomonas syringae* pv. *syringae* 61 $HrpZ_{Pss}$ Protein," Plant Journal 9(4): 431-439 (1996) ("Strobel") (attached to Supplemental Wei Declaration as **Exhibit 14**) (Supplemental Wei Declaration ¶ 19).

The Supplemental Wei Declaration demonstrates that the hypersensitive response elicitor from *Xanthomonas campestris* pv. *pelargonii* (i.e., HreX) induced disease resistance in tomato against bacterial wilt caused by *Pseudomonas solanacearum*, as well as disease resistance in tobacco against tobacco mosaic virus (Supplemental Wei Declaration ¶¶ 21-23).

Thus, application of a broad range of hypersensitive response elicitors to plants have been shown to induce disease resistance.

Hypersensitive response elicitors from *Erwinia amylovora* and *Pseudomonas syringae* pv. *syringae* are known to enhance plant growth. See Examples 1 to 24 of U.S. Patent No. 6,277,814 to Qiu et al. ("Qiu") (attached to Supplemental Wei Declaration as **Exhibit 15**), which showed that treatment of plants and plant seeds with HrpN from *E. amylovora* induced plant growth enhancement in species of tomato, potato, raspberry, and cucumber (Supplemental Wei Declaration ¶ 20). This is further demonstrated by the data presented in the Declaration of Zhong-Min Wei Under 37 C.F.R. § 1.132 filed September 27, 2002 ("Wei Declaration") and in the Supplemental Wei Declaration, which show that treatment of tomato seeds with the hypersensitive response elicitors from *Pseudomonas syringae* pv. *syringae* (i.e., HrpZ) and *Xanthomonas campestris* pv. *pelargonii* (i.e., HreX) independently resulted in significant increases in plant height over that of the buffer treated control (Wei Declaration ¶¶ 9-11; Supplemental Wei Declaration ¶¶ 24-26). Evidence has also been presented in this case showing that both *Arabidopsis* and cotton plants grown from seeds harvested from plants that had been transformed with a DNA molecule encoding the HrpN hypersensitive response elicitor from *Erwinia amylovora* exhibited enhanced growth (Wei Declaration ¶¶ 5-8). Thus, application of a broad range of hypersensitive response elicitors has been shown to enhance plant growth.

Hypersensitive response elicitors from various sources have been shown to be capable of imparting resistance to heat stress, chemical stress, salt stress, and stress caused by calcium deficiency. For example, the Supplemental Wei Declaration presents experimental evidence showing that treatment of plant seeds with a hypersensitive response elicitor of *Erwinia amylovora* ("harpin$_{Ea}$") in non-infectious form can impart salt stress resistance plants grown from treated seeds (Supplemental Wei Declaration ¶¶ 27-30). In addition, the Supplemental Wei Declaration shows that treatment of plants with HreX from *X. campestris* pv. *pelargonii* also induces various forms of plant stress resistance. For example, the treatment of corn with HreX was shown to induce chemical stress resistance and the treatment of lima beans with HreX was shown to induce salt stress resistance (Supplemental Wei Declaration ¶¶ 31-35).

From all of the foregoing literature and cited data, there is ample evidence to show that hypersensitive response elicitors from a limited number of plant pathogens are an art-recognized class of proteins which achieve a variety of common beneficial effects in plants. In view of the similarity amongst the hypersensitive response eliciting proteins of different pathogenic species, as well as the similarity of the genes encoding the various hypersensitive response elicitors, these elicitors clearly constitute an art recognized class of compounds. On this basis alone, it is clear that the restriction requirement made in this case is improper. Further, having demonstrated that plant disease and stress resistance is imparted and plant growth is enhanced with a wide variety of hypersensitive response elicitors, one of ordinary skill in the art would expect a new effect achieved with one hypersensitive response elicitor to be shared by other such elicitors.

The PTO has also failed to satisfy its burden of demonstrating that the claimed inventions are unrelated. In the September 7, 2001, office action, the PTO recited as the appropriate test: "[i]nventions are unrelated if it can be shown that they are not disclosed as capable of use together and they have different modes of operation, different functions, or different effects" (citing MPEP §§ 806.04, 808.01) (emphasis added). However, the PTO has failed to demonstrate that the inventions actually have different modes of operation, different functions, or different effects. Clearly, the function of each claimed method is to enhance plant growth; thus, each claimed method has the same function and effect. The PTO has asserted that since different hypersensitive response elicitors have different nucleic acid coding sequences, they must involve different modes of operation. However, the PTO provided no evidence to support this assertion. In light of the above demonstration that hypersensitive response elicitors are an art-recognized class of compounds, the opposite seems to be true – i.e., their mode of action is similar. In any event, since the PTO has clearly failed to carry its burden, the restriction requirement is improper and should be withdrawn.

Given the presence of generic linking claims and a sub-genus Markush group which recites the use of nucleic acid molecules encoding hypersensitive response elicitor proteins or polypeptides derived from different plant pathogens, the proper course in the instant application is to include the claimed inventions of Groups I-VIII together in a single group, requiring only an election of species at this time. See MPEP § 808.01(a). This procedure is proper for applications containing generic or Markush-type claims, which is exactly the situation here.

Finally, applicants question the proposed classification of each group asserted in the outstanding restriction requirement. It appears the PTO recited different class/subclass designations for each group of invention as support for the proposition that examination of more than one group would constitute a burden to the PTO. In reviewing the on-line PTO classification manual, however, it appears (in most instances) each group of invention would be searchable under almost all of the cited class/subclass designations. There appears to be no basis for including one group in a particular class/subclass while excluding others.

As one example, the PTO indicated that the invention of Group I, limited to the claimed method where the hypersensitive response elicitor is derived from *Erwinia chrysanthemi*, would be classified in class 800, subclass 278. The definition for class 800, subclass 278, recites: "METHOD OF INTRODUCING A POLYNUCLEOTIDE MOLECULE INTO OR REARRANGEMENT OF GENETIC MATERIAL WITHIN A PLANT OR PLANT PART: This subclass is indented under the class definition. Method for insertion of polynucleotide molecules into, or rearrangement of genetic material within a plant cell, wherein said cell is part of, or regenerated into, a plant or plant part." Based on this definition, there does not appear to be any basis for distinguishing Group I from any of Groups II-VII. Therefore, this class/subclass should be searched for each group of invention.

As another example, the PTO indicated that the invention of Group II, limited to the claimed method where the hypersensitive response elicitor is derived from *Erwinia amylovora*, would be classified in class 536, subclass 23.7. The definition for class 536, subclass 23.7 recites: "Encodes a microbial polypeptide: This subclass is indented under subclass 23.1 [DNA or RNA fragments or modified forms thereof]. Compounds which are DNA fragments which encode specific microbial polypeptides." Based on this definition, there does not appear to be any basis for distinguishing Group II from Groups I and III-VIII in this regard. Therefore, this class/subclass should be searched for most groups of invention.

In yet another example, the PTO indicated that the invention of Group III, limited to the claimed method where the hypersensitive response elicitor is derived from *Pseudomonas syringae*, would be classified in class 800, subclass 298. The definition for class 800, subclass 298 recites: "Higher plant, seedling, plant seed, or plant part (i.e., angiosperms or gymnosperms): This subclass is indented under subclass 295 [plant, seedling, plant seed, or plant part per se]. Subject matter wherein the plant, seedling, plant seed, or plant part is a higher plant, i.e., an angiosperm or gymnosperm, both of which produce seeds." Based on this definition, there does not appear to be any basis for distinguishing Group III from Groups I-II and IV-VII. Therefore, this class/subclass should be searched for each group of invention.

As a further example, the PTO indicated that the invention of Group IV, limited to the claimed method where the hypersensitive response elicitor is derived from *Pseudomonas solanacearum*, would be classified in class 800, subclass 288. The definition for class 800, subclass 288 recites: "Nonplant protein is expressed from the polynucleotide: This subclass is indented under subclass 278 [see above definition]. Method wherein the polynucleotide encodes a polypeptide not originating from a plant." Based on this definition, there does not appear to be any basis for distinguishing Group IV from Groups I-III and V-VII. Therefore, this class/subclass should be searched for each group of invention.

In yet another example, the PTO indicated that the invention of Group V, limited to the claimed method where the hypersensitive response elicitor is derived from *Xanthomonas campestris*, would be classified in class 435, subclass 468. The definition for class 435, subclass 468 recites: "Introduction of a polynucleotide molecule into or rearrangement of a nucleic acid within a plant cell: This subclass is indented under subclass 440 [PROCESS OF MUTATION, CELL FUSION, OR GENETIC MODIFICATION]. Processes of inserting polynucleotide molecules into or rearranging genetic material within a plant cell." Based on this definition, there does not appear to be any basis for distinguishing Group V from Groups I-IV and VI-VII. Therefore, this class/subclass should be searched for each group of invention.

In a still further example, the PTO indicated that the invention of Group VI, limited to the claimed method where the hypersensitive response elicitor is derived from *Phytophthora*, would be classified in class 435, subclass 419. The definition for class 435, subclass 419 recites: "Plant cell or cell line, per se, contains exogenous or foreign nucleic acid: This subclass is indented under subclass 410 [Plant cell or cell line, per se]. Subject matter wherein the plant cell or cell line has been transformed by the insertion of nucleic acid which is either exogenous or foreign to it." Based on this definition, there does not appear to be any basis for distinguishing Group VI from Groups I-V and VII. Therefore, this class/subclass should be searched for each group of invention.

In a final example, the PTO indicated that the invention of Group VII, limited to the claimed method where growth enhancement is achieved by topical application of a hypersensitive response elicitor to transgenic plants transduced with a hypersensitive response elicitor encoding gene. Applicants submit that this group is generic to use with a hypersensitive response elicitor encoding gene from any source. Further, the subject matter of Group VII would be classified in class 800, subclass 290 having the following definition: "The polynucleotide alters plant part growth (e.g., stem or tuber length, etc.) This subclass is indented under subclass 278 [see above definition]. Method wherein the polynucleotide

causes the plant or plant part to be larger or smaller or to grow at a faster or slower rate than in the absence of said polynucleotide." Based on this definition, there does not appear to be any basis for distinguishing Group VII from Groups I-VI. Therefore, this class/subclass should be searched for each group of invention.

In view of the foregoing, applicants respectfully submit that the restriction requirement is improper and, therefore, should be withdrawn in its entirety. Applicants would agree, however, that an election of species requirement may be appropriate with respect to the source of the hypersensitive response elicitors. In the event that such an election of species requirement were to be imposed, applicants provisionally elect hypersensitive response elicitor proteins or polypeptides derived from *Erwinia amylovora*.

Applicants believe that no fee is required for filing this petition. If additional fees are required, however, the Commissioner is hereby authorized to charge any fees to Deposit Account No. 14-1138.

Respectfully submitted,

Date: January 21, 2003

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